THE
LARYNGOSCOPE.

VOL. XXIX. ST. LOUIS, SEPTEMBER, 1919. No. 9.

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

PRESENT STATUS OF THE TEACHING OF THE DEAF.*

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The origin of the Society of Progressive Oral Advocates is an humble, though interesting, bit of educational history. At the close of the summer session of 1917, at the Central Institute for the Deaf, a number of graduates and post-graduates of our teachers' training classes suggested the formation of an alumni society to weld into a closer union the working strength and the personal element which had been developed since the establishment of these classes at the Central Institute. In the informal discussion preceding this organization, it was found that the plan and scope of work projected for this alumni was of more formidable proportions than such a limited organization could expect to successfully carry out. The idea was then suggested that the constitution and by-laws be framed along broader lines to meet the requirements of a national society in which could be included all of the educational influences and interests contributory to the progress and advancement of Pure Oralism in its every phase and angle.

The name, "Progressive Oral Advocates," was chosen after much deliberation. "Progressive," as it represents ideas and the modern application of energy; "Oral," as it indicates the funda-

*President's address before the Second Convention of the Society of Progressive Oral Advocates held at St. Louis, Mo., June 23-25, 1919.

mental principles for which this organization stands and as it clearly defines the character of the work that may emanate there; "Advocates," a comprehensive word, including in its broad scope the members of the teaching and medical profession, psychologists, social service workers, the parents and friends of the defective child and all other earnestly inclined men and women who by co-operation in a large organization with a definite purpose may be found throughout the land to add their energy and influence in the upbuilding of the splendid cause for which the Society of Progressive Oral Advocates stands.

The organization meeting of this society, held in St. Louis in June, 1918, exceeded even our fondest expectations. The interest with which this call for organization was made by the best teachers throughout the land, the numerical strength with which the first meeting was attended, the uniformly high scientific quality of the papers and demonstrations presented at the meeting, and the favorable comments of every delegate and member attending this first meeting, bespeaks for this organization, strength, success, influence, and accomplishment. The lessons of the war, when the American nation found themselves involved in the ramifications and numerous problems of the World's War, it soon became evident that the phase of special education and special science which formed so intimate a part of the work of this organization would be called upon for a practical demonstration and for a utilitarian application of principles. Last week I attended the seventieth annual convention of the American Medical Association, where the medical and surgical aspects of the war were discussed in their many phases. It was the consensus of opinion that the most important developments in the medical sciences had been made in preventive medicine, sanitation, and reconstruction. In preventive medicine was included the tremendous problems of coping with tuberculosis, the management of epidemics, the morale and strict military regulations to protect soldiers against the spread of sexual diseases, the investigation of the etiology of the awful epidemic of "flu" that swept the nation from coast to coast and from one army corps to another—vital questions which will engage the energies and resources of the medical profession for many decades to come.

Reconstruction, as it applies to war issues, has proven to be one of the most important phases of practical service to every soldier in the field. The unusual character of modern warfare—

trenches, high explosives, gas bombs, hand grenades, and close-range combat, have produced a tremendous preponderance of wounds in the head. Head wounds, injuries, and mutilation of the face, detonations of heavy artillery, and its subsequent deafness, gas bomb explosions and ensuing blindness—all call for the greatest tax on skill and resource of the surgeon. But it required more than the surgeon's skill to reconstruct the crippled soldier. You, as experienced workers among the deaf and those defective in speech, have been called on for an important contribution of your work in the rehabilitation and re-education of the injured soldier. With its usual far-sightedness, our government demonstrated its preparedness in this as well as in every other field. Shell-shock and shell concussion caused numerous cases of deafness. Explosives and close-range fighting, with subsequent mutilation of lips, cheeks, mouth, nose, jaws, and neck, necessitated, after convalescence, re-educational measures by which the functioning machinery of speech, of hearing, of sight could be re-adjusted or rehabilitated.

A comparatively small group of experienced teachers quietly and effectively carried out their practical work in re-education by Lip-reading and Corrective Speech at the U. S. A. General Hospital Number Eleven at Cape May, and the enthusiasm and interest with which the members of the American Otological Society in Annual Convention at Atlantic City last week appreciated a demonstration of the work done on a group of deaf and defective speech soldiers should make you all feel proud of the cause and the profession in which you are engaged.

At the meeting of the Society of Progressive Oral Advocates last year, Mr. John Dutton Wright offered a resolution which was unanimously adopted that steps be taken to present arguments in favor of making impaired hearing in children reportable and to endeavor to have such an act passed by the legislature of each state and by the nation. By such legal and protective measure, it might be made possible to safeguard the interests of the deaf child in every state and to keep in touch with his educational opportunities as readily as with the normal child.

While the committee of this society to which has been entrusted this important resolution has not met officially to formulate its plan of work, it has not been idle in the development of this important problem. I have the honor to report that this matter was discussed and enthusiastically received at the meet-

ing of the Section on Otology and Laryngology of the American Medical Association last week in Atlantic City, and that a permanent committee, consisting of Dr. Charles W. Richardson, of Washington, Dr. E. L. Kenyon, of Chicago, and Dr. Max A. Goldstein, of St. Louis, has been officially appointed to take charge of these questions affecting the health and the education of the child defective in hearing or speech. This committee has already been assured the support of the American Medical Association through its representative council as soon as resolutions have been drawn up and ratified by the Section on Otology and Laryngology.

In outlining some of the progressive measures with which this society could creditably identify itself, I referred last year to a committee on Survey and Standardization of all schools for the deaf. The continuance of the war has handicapped the work of this committee and my own participation as chairman of this committee has been made almost impossible because of my active war service. The next step in the deliberations of this body will be taken, however, after hearing the paper on "Standardization of Schools for the Deaf," by Mr. R. D. Johnson of the Indiana State School for the Deaf. It will be recalled that a similar committee had been appointed several years ago by the American Association of Superintendents and Principals of Schools for the Deaf, and Mr. Johnson's report will, no dobut, throw some light on the work of this previously appointed committee.

The question is a vital one and concerns every earnest, honest oral advocate. Standardization in educational measures and educational equipment as applied to the deaf child is even more important than similar standardization for the education of the normal child.

The ultimate and general acceptance of Oralism depends largely on the qualification of oral teachers and on the results of their work. Poor or improperly-qualified oral teachers will do more toward handicapping the ultimate universal establishment of oral teaching than any other factor in this work. The invincible weapon with which to contend against the manualist, the signer, and the combiner is not argument, not criticism, not politics, but the deaf child intelligently trained in good fluent, comprehensible normal speech. Oralism must succeed, not by antagonism and small politics, but by actual practical evidence of accomplishment.

On my way to Atlantic City I visited Mt. Airy. What an inspiration it is to see this beautiful group of buildings in a most charming park and forestry setting, the contribution of the State of Pennsylvania to the Education of the Deaf. What a stimulation to meet that kindly and able veteran, Dr. Crouter, in his home surroundings and to realize that through his indefatigable efforts over a period of many years, he had finally succeeded in establishing a system of exclusively oral instruction in this large State school. Where this pioneer in Oralism has blazed the way, surmounting one handicap and difficulty after the other, surely others could follow. Oralism has gained many adherents since the day when Dr. Bell and Dr. Crouter and Dr. Yale first championed its cause. There are better opportunities for development, there are more legislators with open minds, there are more and better qualified oral teachers, there are many parents and friends of deaf children who have been firmly convinced of the value of oral training. It seems to me that the path is clear with the exception of a few political and financial obstacles and every state and every community should soon find it possible to espouse this cause. It needs intelligent direction; men's minds are receptive today to all forms of progress and you, advocates of Oralism, should be the guiding influences through which Oralism may find a more intelligent and enthusiastic consideration in every state and community in the land.

Instead of attempting to force the issues for Pure Oralism by cut and dried legislative measure, lobbying bills, which are passed and then forgotten, speeches against signers and combiners, let me urge you to make every issue and every argument one of human interest. There is no better illustration of my point than my recent experience while in service.

During the past winter I was stationed at the Base Hospital in Camp Dodge, Iowa. One day my telephone rang, and I was greeted by a lady in Des Moines, who told me that the Executive Committee of the Society of Friends of the Deaf in the State of Iowa were seriously considering the drafting of a bill to be presented to the legislature of the State of Iowa, asking for an appropriation of $50,000 for a pavilion at the State School in Council Bluffs, in which young, orally trained children could be segregated and supervised. I was invited to meet this committee. I attended the meeting and heard the substance of the bill. I was asked for an expression about this bill. I frankly

told the committee that this bill was so impersonal in character that it might receive but scant consideration at the hands of the State Legislature. I offered, as a substitute, to make a direct appeal to the Joint Committee on Education of the Iowa Senate and House, by facing them with the actual documents in evidence. After brief preliminaries, the nine kiddies of the recently organized Oral class in the city of Des Moines, together with their teacher, met me in the committee chambers of the Iowa capitol and we proceeded to demonstrate practically to the sturdy legislators of this good state what speech and lip-reading and oral training actually meant. Suffice it to say that our deaf kiddies won their way to the heart and receptive mind of every committeeman and the appropriation was voted without delay.

What is possible in the State of Iowa is possible in every other State of the Union. The appeal and influence of the deaf child, carefully trained to good speech and fluent lip-reading, is a human appeal—an understandable thing, to a group of legislators, to a committee on educational work, to a school board, to intelligent laymen the world over. Such a human appeal and practical example must be productive of untold good results. This practical form of demonstration is the only way that I have found to reach a responsive chord in nearly twenty-five years in my efforts to convince the members of the medical profession and of my own speciality as to the real merits and value of oral work. The interest of the medical profession is gradually being awakened in this big work. Doctors are beginning to realize that they owe the community something more than personal service in their own chosen field. Their training and experience qualifies them to act in advisory capacities in the development of special educational work. The responsibility of the doctor, the teacher, and the citizen at large to the defective child—defective in sight, hearing, speech, limb, or mind, is as great as the responsibility to the normal child. The war has taught us another great lesson—co-operation. We are more keen to the necessities of upbuilding the community, and the state; we realize the importance of better and more dependable citizenship; we have been convinced of the importance of better educational measures, for it makes better soldiers, better parents, better citizens, better service to the community.

Legislators, educators, professional and laymen have been convinced that more time, energy, money and brains must be spent in the development of the child.

A significant illustration of this fact occurred to me during a recent visit East when I was an invited guest at the annual banquet of the Round Table for Corrective Speech at which our own fellow delegate, Dr. Frederick Martin of New York City, presided. Gathered for the annual luncheon were 450 teachers actively engaged in special instruction in the City of New York, and about twenty invited guests. For three hours the eight invited speakers on this program addressed this intelligent audience on the problems of Corrective Speech and on ways and means for better speech in the City of New York. It must be a source of gratification to the few organizers of this Corrective Speech movement in the East to know that in the course of two or three years such a large and splendid working force could be gotten together; it must be an index to everyone of us of the necessity and importance of this movement.

At a survey made of the City of St. Louis about a year ago, it was found that over two thousand of the children attending the public schools of this city had some form of defective speech of varying degree. Our school authorities have not yet successfully disposed of this question. The reasons are evident; lack of funds, lack of information on these questions on the part of the school board, and lack of sufficient and properly qualified teachers. Money, enlightenment, and teaching efficiency, there are the three grand bulwarks of educational progress everywhere.

Our contribution and our progress may be sharply defined. On *our* shoulders rests the responsibility of producing sufficient and properly qualified teachers for schools for the Deaf and for Corrective Speech. Produce the teaching machinery and show the end results of efficient teaching methods to the proper authorities in every community and in every State and enlightenment must follow. With enlightenment comes the inspiration to the intelligent upbuilding of the community; with enlightenment come the ways and means of judicious expenditures of moneys; with enlightenment comes the desire for larger appropriations, when the purpose and the result to which such appropriations are applied have been recognized.

BONE CONDUCTION OF SOUND IN CETACEA AND ITS RELATION TO INCREASED BONE CONDUCTION IN HUMAN BEINGS.*

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One of the most important symptoms of chronic middle ear catarrh is the relative and absolute increase of bone conduction found in that condition. Many of the tests on which the differential diagnosis depends, such as Weber's, Schwabach's, etc., are based upon it; yet nowhere is there agreement among otologists as to its cause. There appears, moreover, to have been done little experimental work with a view of explaining it.

Several theories are advanced concerning the phenomenon. First is that of Bezold, that in consequence of the changes within the middle ear, although the mobility of the conducting chain of ossicles may be interfered with, yet the tensing of the fibers of the ligamentum annulare renders possible an easier conduction of sound waves coming through the bone to the foot plate of the stapes. A second explanation is that the sound waves coming through the bone are transmitted directly to the structures within the cochlea. Still another is that under conditions of closure of the external auditory meatus, the air within the tympanum receives the sound vibrations from the bony walls of the cavity and transmits them increased in force to the foot plate of the stapes, the closed cavity acting as a resonance box.

In making some studies on cetacea, it occurred to the writer that in the anatomical structure of the ears of these animals we might find an explanation of the increased bone conduction of sound at least under some circumstances. Lest anyone question the fact of whales having hearing on account of our having no experimental proof, I shall here call attention to two evidences of it. Whalers and others having to do with these animals all testify as to their ability to hear water-borne sounds. In hunting the animals no precaution need be taken as to air borne sounds such as conversation. Sounds which would be carried through the water, however, such as splashing of oars,

*Offered as Candidate's Thesis for the American Laryngological, Rhinological and Otological Society, June 19, 1919.

or blows against the sides of the boat, must in every way be avoided.

The second evidence of acuity of hearing in cetaceans lies in the structure of the ear itself. The cochlea, the organ of sound perception, is largely developed, presenting two and a half turns. If we contrast this condition with the complete disappearance of the olfactory organs and nerve in some cetaceans, the odontocetes, and their great reduction in others, the mystacocetes, we may fairly argue that as the lack of use of the sense has brought about disappearance of the organ in the one case, its continued use has caused its retention in the other.

Considering then the ability of cetaceans to hear as established, we may go on to consider the anatomical structure of the ear. The material on which this study is based consists of a foetus of Kogia breviceps at term and several specimens of Tursio tursiops for the odontocetes, and a foetus of Balaenoptera borealis, 108 cm. long, for the mystacocetes. I have also had at my disposal a considerable number of odontocete skulls.

The arrangement of the tympanic and periotic bones in cetaceans is so peculiar and unique among animals that a few words must be devoted to them.

In odontocetes the periotic, corresponding to the petro-mastoid of human anatomy, consists of a centrally placed ball-like mass of bone, from which project, craniad and caudad, two solid masses. The first of these corresponds to the human tegmen tympani. The second has been named by Denker,[1] the "processus tympanicus." The central mass itself contains a minute canalicular portion and a very well developed cochlea, containing two and one-half turns. The usual foramina on tympanic and cranial surfaces can be made out.

The ostympanicum has the form of a semi-cylindrical sea-shell with a thin, irregular lateral border and a massive, rolled over mesal border. The portion corresponding to the human tympanic ring is merely a small notch in the lateral border. To the cranial margin of this notch the long process of the malleus is firmly fused. Between the two bones is enclosed a comparatively small tympanic cavity, ventral of which is a large space enclosed in the bulla. From the caudal region of the tympanicum projects a massive "processus perioticus" (Denker, op. cit.) which articulates with a corresponding process of the periotic, and in some forms synostoses with it. In adult animals this process further develops into an enlargement known as the tym-

pano-mastoid, which appears largely on the surface of the skull in a notch between the squamosal and exoccipital. The presence of this large mass, an outgrowth of one of the bones related to the organ of hearing, should be noted, as it had in all probability much to do with the ability of the animal to hear.

The two bones, tympanic and periotic, are united in one other place by bony union, a small area farther rostrad on the outer border of the bulla. Elsewhere they are separated by fissures of varying width. Together they lie entirely outside the skull in a space bounded by the basi-occipital, the exoccipital and the squamosal, and they are buried in a mass of cavernous tissue enclosing blood and air spaces.

Fig. 1—Periotic and tympanic bones of Zipluis Cavirostris.

In the mystacocetes the arrangement of the bones is much the same as described for the odontocetes. The tympanic forms a semi-cylindrical bulla, having a similar over-rolled internal and irregular external edge in a deep groove of which is set the membrane tympani. To one marked difference attention should be called. The process of the tympanic referred to as the tympano-mastoid is absent in the mystacocetes. Its place is taken by a true large mastoid process, a portion of the periotic, which appears on the surface of the skull between exoccipital and squamosal bones just as in other mammals. Tympanic and periotic have two areas of bony union, slight in extent. The two together are only loosely joined to the other

bones of the skull, and in dried preparations can be picked out without much difficulty. In the mystacocetes also the malleus forms a firm bony union with the tympanic.

To certain features which the two orders of cetacea have in common I should now like to call attention. Both expose on the surface of the skull a large bony process which is capable of receiving sound vibrations impinging on it through the water.

According to Van Kampen,[6] it is the os tympanicum which forms the main mass of the mastoid process in mystacocetes as in odontocetes. In this young specimen the true mastoid alone is present. In both we find fusion of the malleus to the exter-

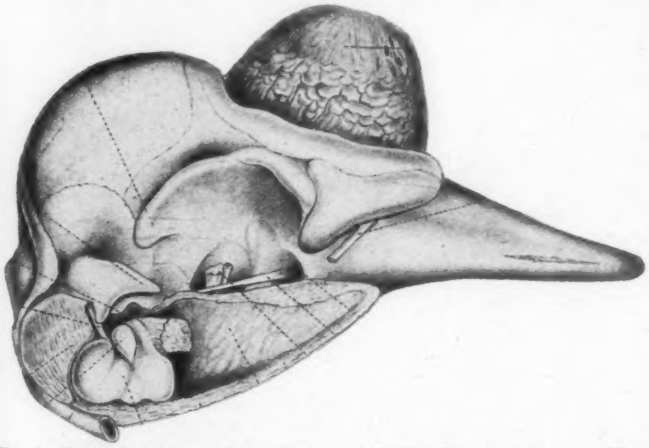


Fig. 2—Kogia Breviceps. Lateral view of skull with exposure of auditory region.

nal border of the tympanic bone. In both, the tympanic and periotic bones have a double bony union, their margins elsewhere allowing communication of the tympanic cavity and surrounding parts. Finally, in both, the conjoined bones of hearing are free from the other bones of the skull.

The following description of the auditory apparatus in Kogia is taken from a work on that subject previously published by the author.

The external auditory orifice is a minute orifice situated caudad and ventral of the eye. There is no trace of an auricle, nor in the specimen examined were any distinct muscles related to the margins of the orifice found (Hanke).[2] The meatus passes mesad and rostrad in a groove of the glenoid process of the squamosal, to terminate at its attachment to the os tym-

panum. The walls of this canal are firm, fibrous structure, of moderate thickness and without cartilage as far as could be ascertained. The mouth of the canal and at first its lumen are so minute as to forbid probing. Approaching the tympanum, the lumen expands in a trumpet-shaped manner, though still of insignificant size.

The tympanic membrane is an exceedingly thin sheet, faces dorso-mesad and is attached to the caudal and ventral borders of the notch it occupies. Rostrad, it passes beyond the border to find attachment in a groove on the ental surface of the sigmoid process in the manner described by Denker for Phocaena. Dorsad it is attached to the fibrous band which bridges over the space between the anterior and posterior conical proc-

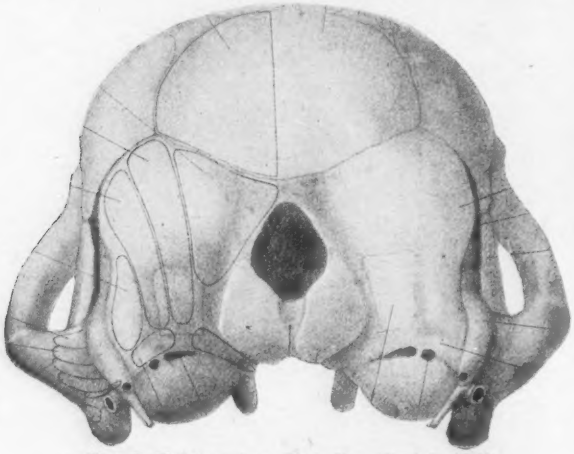


Fig. 3—Balaenoptera Borealis. Foetal skull.

esses. As far as could be ascertained, the surface of the membrane is slightly concave ectad, as in other odontocetes and in young baleen whales. Its inner surface is connected to the malleus by a triangular fold of mucus membrane.

The tympanic cavity, lying between the periotic (dorsad) and the broad rolled-over mesal border of the os tympanicum (ventrad) is a comparatively limited space, owing its dorso-ventral extension chiefly to the hollowing of the tympanic surface of the periotic. It has, however, communications with extensive air spaces lying beyond the borders of the limiting bones.

The cavity, in its natural state, is occupied by a mass of thick tissue which lines its walls and nearly fills it, in this manner

concealing the ossicles. Wherever the os tympanicum and periotic fail to meet it fills in the space between them, and is continuous with the mass of cavernous tissue in which the whole complex lies buried. So it, too, is undoubtedly of cavernous nature. It fills in the hollow of the bulla and extends into the excavation beneath the inverted mesal border of that structure, also into the excavation of the tympano-mastoid. This cavernous tissue is probably of importance in regulating the pressure within the middle ear when the animal is submerged.

On the removal of the cavernous tissue the structure of the middle ear comes into view. The inner wall (or dorsal wall, as it is here) presents, caudad, a short rostro-caudal groove bordered by prominent ridges, the channel for the seventh nerve.

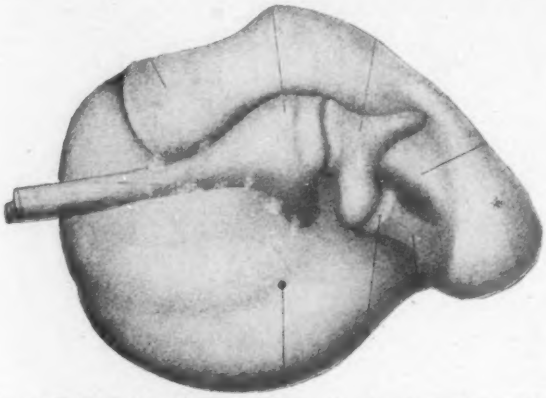


Fig. 4—Balaenoptera Borealis. Left otic capsule, external view.

Mesad of the groove is a ridge which is overlain by the stapedious muscle, and, just at its termination, the stapes is firmly secured but not ankylosed in the fenestra ovalis. Rostrad of this is a large depression occupying half the width of the tympanic surface of the bone. When the various parts are in their natural position this depression is occupied by the head of the malleus. Jutting underneath the orifice of the ductus fallopii is a prominent spicule, against which rests the short process of the incus. No tensor tympani muscle was discovered though a groove for one has been noted (Schulte)[3] in the position in which we should expect to find that muscle. It is of interest to state here that in another odontocete (Tursiops) a distinct tensor tympani muscle has been found by the author—a fact not

hitherto noted and by some specifically denied (Denker). It is here much less defined than in baleen whales.

The ossicles, in their general form, do not differ from the usual mammalian type. The malleus has a proportionally large head, which lies in the depression in the periotic already described. The manubrium mallei is fused to the os tympanicum along the border of a narrow cleft in its lateral margin. The usual saddle-shaped articulation unites malleus and incus. The latter bone is distinguished by the fact that the two processes are of equal length, although the processus longus is the thicker. The processus brevis, at its tip, meets a small tubercle which juts out from the crista facialis dorsad to the fenestra ovalis. The stapes is not fenestrated and sits firmly in the oval window. The

Fig. 5—Kogia Breviceps. Mesial view of fragment of tympanum with malleus attachment.

Fig. 6— a. b. c.
Kogia Breviceps—(a) Lateral view of incus. (b) Mesial view of incus. (c) Stapes.

non-fenestrated condition is doubtless secondary, as Denker describes small depressions in the stapes of Phocaena which indicate the existence of a fenestra, and the author has observed the same in other odontocetes, namely, Ziphius and Tursiops.

The tympanic opening of the facial nerve lies just above the oval window. At this point the nerve turns sharply dorsad and takes a straight course through the periotic. Thus the geniculate ganglion must lie within the tympanic cavity. This is due to the non-development of that portion of the tegmen tympani which in other mammals (as for instance, man) cover in the facial canal for the latter part of its course in the tympanum. A minute canal for the great superficial petrosal nerve passes rostrad between tegmen tympani and periotic, as it has

been found in Tursiops and Balaenoptera. Since the periotic lies entirely without the cranial cavity the great superficial petrosal nerve nowhere enters the skull.

There are two points of contact and fusion between the os tympanicum and periotic: the anterior is, in this animal, already a bony union; the posterior consists of an exceedingly firm fibrous union of the tympanic process of the periotic to the dorsal surface of the tympano-mastoid. Except for these points, the bones are not in contact and the intervals are filled in by cavernous tissue.

At three places this tissue wall is broken and there the air space of the tympanum communicates with the system of air spaces without. These have been described by Denker for Phocaena as lying in three groups: lateral, caudal, and rostrad. A like arrangement is found in Kogia. The lateral group opens out through the space between membrane tympani and periotic. It here consists of a few small cells. The posterior group communicates with the tympanum by an opening lateral to the facial canal and lies in the space bounded by the tympano-mastoid and the basi-occipital process of the occiput. These cells also are of insignificant size. The anterior group, opening through the gutter of the os tympanicum contains only one cell but the this is of remarkable dimensions. It is an oval space, 3 cm. by 5 cm., which occupies the whole pterygoid fossa. Its mesal wall, as are the walls of all these air sinuses, is made up of a thick pad of cavernous tissue. Its thin outer wall is overlain by the internal pterygoid muscle. Ventrally in the angle of junction of the mesal and lateral walls, a minute open opening allows a probe to pass through the pterygoid notch into the naso-pharynx. Thus this large space is evidently an expansion of the Eustachian tube. It communicates with the naso-pharynx and the intimate relation to its lateral wall of the pterygoid muscle evidently permits of its distension by air and the expulsion of the same.

In baleen whales, as represented by the Balaenoptera borealis examined by the author, the external orifice of the auditory canal is a minute slit caudad and dorsad of the eye. The meatus is very small as in the odontocetes, and at its inner end expands like a trumpet. The drum membrane is a thin oval membrane, slightly concave toward the inner surface, attached by its margin to the tympanic ring. It has relation to the manubrium mallei only through the triangular ligament a fold of mucous membrane similar to that found in odontocetes.

It must be remembered that the above description applies only to an immature animal. In the adult, Beauregard[4] has found that the drum membrane bugles outward into the external auditory meatus like the finger of a glove, and in large part loses its resemblance to the ordinary mammalian drum membrane. In adult Balaenoptera also, the inner end of the auditory canal was found by Lillie[5] to be occupied by a ceruminous plug.

The cavum tympani is a bowl-shaped cavity having dorsal and ventral walls which meet in a sharp angle at their margins. The ventral wall contains in the lateral area the membrane tympani surrounded by the crescentic os tympanicum. The rest of the ventral wall is made up of the fibrous bulla which fills in the space between os tympanicum and the marginal attachment of the bulla.

The dorsal wall of the tympanic cavity is made up of the otic capsule centrally placed, and circumferentially of a ring of fibrous tissue which connects the petrosum to the surrounding bones. As it presents itself after removal of the os tympanicum and bulla, it is seen to be covered by a layer of thick tissue which completely conceals the underlying cartilages and almost fills the cavity. In the outer area of the cavity this structure throws folds about the ossicle and only on its removal can they be examined. The formation of this tissue shows it to be of a cavernous nature (Beauregard).

The tuba auditiva passes from the choana laterad between hamular and vaginal processes of the internal pterygoid and penetrates the wall of the bulla obliquely. Its entrance into the tympanic cavity is at the rostral circumference of the same in the angle formed by the meeting of ventral and dorsal walls. The opening is a crescentic slit capable of valveo-like closure. The tube is very short, merely an oblique passage through the fibrous wall of the bulla. The expansion of its distal end into the scaphoid fossa is not yet indicated.

The ossicles are typically mammalian, their arrangements Meckel's cartilage passes beneath the edge of the tympanic bulla, caudad and slightly dorso-laterad, closely roofed over by the tegmen tympani. Mesad to its shaft is the belly and tendon of the tensor tympani muscle. Within the tympanic cavity, the cartilage expands into a fairly large caput mallei, and forms a mesad projection, the manubrium to the base of which is attached the tensor tympani. A groove in its surface completely encircles the caput, close to the edge of the articulation with

the incus. The border itself expands, thus increasing the depth of the groove.

The incus has a triardiate form. On the well-developed body is a saddle-shaped articular surface for the malleus. The processus brevis is stout and of large size. Its extremity is in contact with the crista parotica, to which it is firmly attached by a ligament. The processus longus, which actually is shorter than the brevis, is also bulky in form. It is directed ventrad to articulate through an os articulate with the apex of the arch of stapes.

The stapes, which are lodged in a deep fossula, does not fill with its foot plate the large fenestra, but is united to its circumference by a rather broad annular ligament. The bone closely agrees with the adult form; its arch is high and narrow, the limbs thick, the foramen small.

Comparing these two forms of cetacea we see that in regard to the absence of the external ear and the small size of the external auditory canal, they agree. The odontocetes lack, to be sure, the plug of cerumein found in the canal of the mystacocetes, but even in their case it cannot be imagined that any sound can reach the drum membrane through that passageway. In both alike the drum membrane is disassociated from the ossicles except for the prolonged "triangular ligament." Denker has satisfactorily demonstrated that no movement can be transmitted from drum membrane to ossicles, and also that movement of the ossicles independent of the os tympanicum is impossible.

The arrangement in the tympanum also shows properties in common. A typical mammalian chain of ossicles is found buried in a mass of cavernous tissue. The air space communicates with a series of surrounding air spaces through the intervals between os tympanicum and os petrosum. Of these air spaces, one, the cranial, is formed by a huge expansion of the Eustachian tube. All are capable of inflation from the pharynx. The comparatively loose attachment of the bones of hearing to the rest of the skull is a condition found in all whales. Finally attention is called to the presence on the surface of the skull of a large bony process coming off from the os tympanicum in the case of odontocetes, from the os petrosum in the mystacocetes.

What conclusions then can we draw from the anatomical structure as to hearing in cetaceans? We have presented to us an auditory apparatus, typically mammalian, designed to receive air-borne sounds modified to one designed to receive

water-borne sounds. The external meatus has been practically closed, the drum membrane fixed and the ossicles rendered immovable through fusion of the malleus to the os tympanicum. Sounds are evidently transmitted to the cochlea through the solid tissues of the head. The possibility of this is increased because in both forms the auditory bones themselves present on the surface of the skull a considerable bony process. In both, moreover, the periotic and tympanic are but loosely connected to the other bones of the skull. Thus they can receive only such sound waves as impinge directly on themselves. Since the malleus is firmly fused to the tympanic it would share the vibrations of that bone, transmitting them through the other ossicles to the oval window. This is probably the explanation of hearing in cetacea, that sound waves impinging either on the tympano-mastoid in odontocetes, or the true mastoid in mystacocetes, are transmitted through the chain of ossicles to the cochlea, the endolymph being put into motion through the foot plate of the stapes as in other mammals.

Some authorities maintain that the sound waves reach the receptive organs in the cochlea directly through the walls of the periotic bone. In this connection it is important to recall that whereas such vibrations might provide for a vague perception of sound, they would hardly provide for the interpretation of sound which we are probably justified in thinking depends on an orderly succession of waves in the endolymph from fenestra ovalis to fenestra rotunda. This could be provided for only by vibrations coming through the foot plate of the stapes.

It will now be further recalled that the tympanic and periotic are surrounded by numerous cells capable of distension with air. This is undoubtedly set into motion by the vibrations of the bones of hearing and we may suppose that these air cavities form a kind of sounding box, which increase the sound waves. The air cells then are an important aid in hearing.

We may now consider the light which the mode of hearing in cetacea throws on the clinical symptom of increased bone conduction in deafness due to disease of the middle ear. It will be seen at once that any change which would connect the ossicles more firmly to the tympanic ring, approaching the complete fusion found in whales would increase bone conduction. A tense, thickened, rigid drum membrane would do this. We could also accept Bezold's idea that it is a thick, tense ligamentum annulare, transmitting vibrations to the foot-plate of the stapes.

which in some cases explains the phenomenon under discussion. This would be the condition following a radical mastoid operation. Another theory may now be mentioned. When the external canal is lightly blocked, bone conduction is increased. This is explained by conceiving the air filled cavity with bony walls to be a sounding box. This is just what we have present in cetacea. In this connection it may be said that the variation in the size and number of the cells present in the human mastoid process may very well be an explanation of the variation in the response to tests of bone conduction found in seemingly similar cases of deafness.

We will see then that in cetacea we have all the elements normally present which have been advanced to explain the symptom of increased bone conduction in the diseased ear of human beings, and the anatomical arrangements there found could certainly support the explanations of the phenomenon advanced.

156 East 79th Street.

The Diagnostic Value of Lowered Bone Conduction in Syphilis.

W. H. Goeckerman, R. A. Barlow and J. H. Stokes. *Am. J. Spyhilis,* 1919, iii, 240.

The following conclusions are presented relative to the diagnostic value of lowered bone conduction in spyhilis:

1. The so-called lowered bone-conduction test (reduction in conduction of sound by bone as compared with otherwise normal hearing) was positive in 78 per cent of known syphilitics in the series studied.

2. From the otologic standpoint the test is of value only if a complete hearing test is made.

3. The efficiency of the test varied greatly in different types of syphilis, being at its best in late cutaneous syphilis (100 per cent), latent syphilis (80 per cent), and syphilis of the central nervous system (80 per cent). It had almost no value in osseous lues, and the results in early syphilis were inconclusive (too few cases). A negative Wassermann test combined with a negative bone conduction test is strong evidence of the absence of syphilis.

4. The test agreed with the positive or negative diagnosis of syphilis in 67 per cent, and disagreed in 33 per cent.

5. The test was positive also in 48.7 per cent of patients in whom syphilis could apparently be excluded.

6. On the whole, therefore, the test has only a restricted value as a diagnostic aid owing to its high factor of error.

LAWS OF THE HEARING CENTERS AND THE APPLICATION OF THESE LAWS.

DR. JOHN F. CALLAHAN, Brockton, Mass.

Weber found that a vibrating tuning fork placed upon the skull is much more distinctly heard in that ear the external meatus of which is closed with the finger. The theories advanced why this increased sound perception is produced are:

1. By the increased resonance of the external auditory canal.
2. By reflection of the sound waves transferred through the cranial bones to the air in the external auditory canal, and thence to membrana tympani and ossicles.
3. By the altered tension of the membrana tympani and ossicles.

The last two theories do not stand very strong when we have the same condition existing when the ossicles and membrana are absent.

Bing says that the time perception for the tone of the tuning fork is lengthened in the normal ear if the external meatus is closed with the finger.

Both Weber and Bing are correct as to increased intensity and the lengthened time for the simple reason we hear it longer because we hear it louder and the reason that we hear it louder is because the sound that reaches both internal ears is lateralized to the hearing center on the side that is obstructed or the side that hears it the louder. The reason that the sound is lateralized is because it follows my law of the hearing centers.

When a vibrating tuning fork or watch is placed against the forehead of an individual with two normal ears he is not conscious of hearing the tone or tick in either ear: If then, the external meatus of the right ear is closed he will be conscious of hearing the sound in the right ear and he will also hear it louder.

LAW.

When two sounds which have similar properties except intensity, are transmitted one to each ear, both impulses will pass to the hearing center on that side where the intensity is the greater; and there will be a summation of the two stimuli. We are, therefore, conscious of hearing the sound louder than if the

sound of lesser intensity did not reach the ear, and also if both sounds reach each ear with equal degrees of intensity. The increase due to the summation is not equal to an arithmetical addition but follows Weber's law.

The watch and tuning fork are heard louder when pressed against the teeth than against the forehead, and the intensity is increased when the external meatus is obstructed when they are in contact with teeth and forehead.

By placing a closed funnel or by any other means excluding outside noises from one ear has the same effect as directly obstructing the external auditory canal with the finger or otherwise. The only reasons that I know of why the sound is heard louder when the watch or tuning fork is in contact with the teeth or forehead, and the meatus obstructed, is that by cutting off extraneous sounds on that side, we are able to hear the sound louder in that ear, and this difference is sufficient to cause a lateralization of the sound of lesser intensity that reaches the cochlea of the unobstructed ear, together with the sound of greater intensity in the obstructed ear in the hearing center, on the side that receives the sound of greater intensity. There is a summation of the two sounds which is another factor in increasing the intensity. When we obstruct the meatus of one ear less outside sounds reach the cochlea on that side than does the cochlea of the opposite ear.

That the increase and lateralization of the sound in the obstructive ear is due to the shutting off of extraneous sounds, is shown in the following test: When we shut off the outside sound the tuning fork is heard louder in that ear. Therefore, following my law of the hearing centers, both sounds from the tuning fork pass to each cochlea and then pass to the hearing center on the side where the intensity is the greater, and part of the increase in intensity is due to a summation of the two sounds superimposed in that hearing center. So in these tests instead of obstructing the external auditory meatus of one ear, I increased the outside sounds going to that ear so that that ear would not hear the vibrating fork (which is placed against the teeth), as loud as the ear that was free from the extra outside sound and I found that the sound of the tuning fork and watch was not only increased in intensity but was also lateralized in the ear that was receiving little or none of the extra sound.

Let us see what happens when we increase the sound in one ear when the watch is in contact with the teeth. If we place the

watch against the teeth and a C-1 256 or a 512 vibrating tuning fork a few inches from one ear the tick of the watch is heard louder and if we then obstruct the external meatus of the other ear the intensity is again increased.

With the tuning fork in one ear, the watch cannot be heard as loudly in that ear and the reason that we hear it louder is that we get a lateralization and consequent summation on the other side.

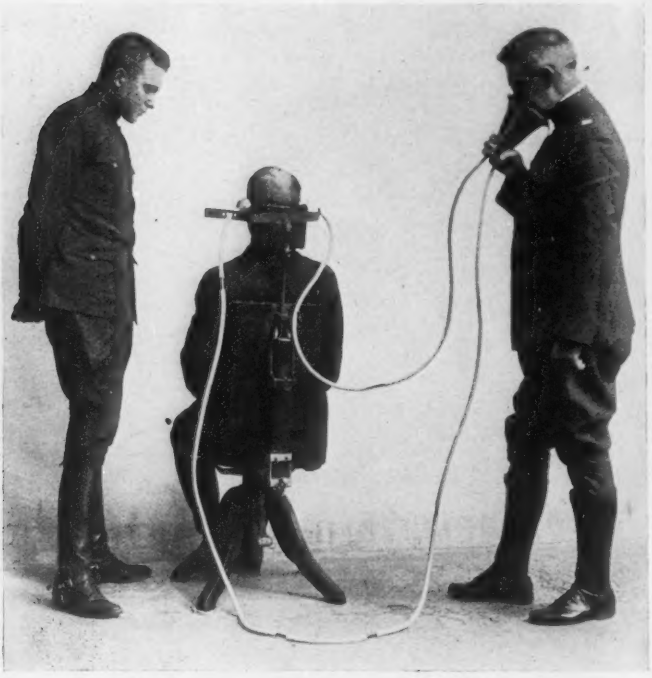


Fig. 1.

But the greatest difference in the increae in intensity is found when the meatus is obstructed while the fork is still vibrating at the opposite ear. With the watch or tuning fork in contact with teeth or forehead and then obstructing both external meati, there is considerable increase in the intensity of the sound, but the sound is not lateralized and the increase is due to excluding outside sounds.

Obstruct the external meatus with an electric noise apparatus (muffle the buzzer, as it makes too great a noise for the

test), place a C-1 256 or a C-2 512 tuning fork against the teeth. The fork is heard in the opposite ear that the noise apparatus is in contact with. Then stop the noise apparatus but keep the meatus obstructed with it and the sound of the fork is clearly noted to pass over to the obstructed side, which it did not do when that ear was receiving the vibrations from the noise

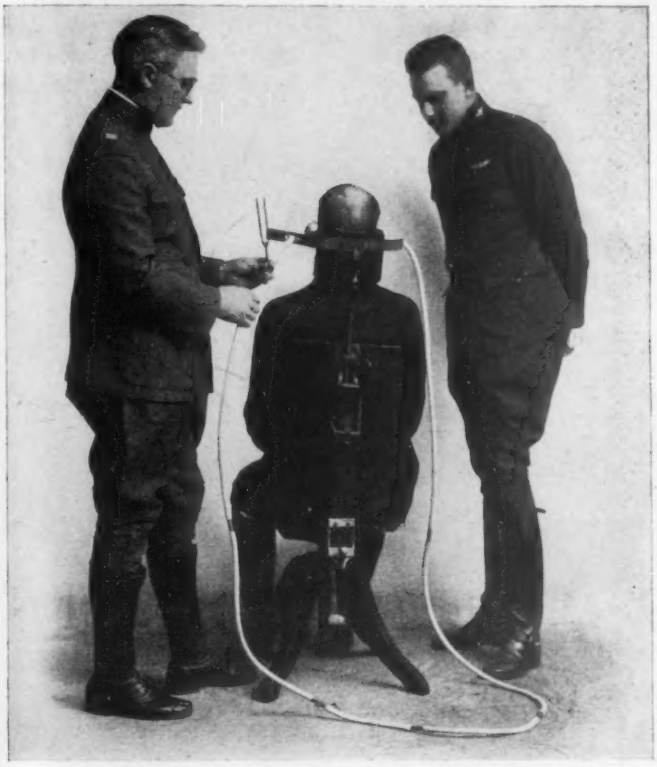


Fig. 2.

apparatus while its canal was obstructed. This shows that a plugging of the meatus aids in the lateralizing and intensifying the sound in no other way than by cutting off outside sounds.

There is no such a thing as increased bone conduction. It is purely and simply a matter of hearing a sound louder when other sounds are cut off from one ear and not from the other ear, together with the lateralizing in one hearing center and the subsequent summation. We all know that we can hear a watch

tick or a whisper in a sound-proof room, that we could not hear in a place where there is considerable noise. In obstructing the meatus the result is just the same whether it is accomplished by disease affecting the ossicles or drum head, by plugging the external auditory canal with finger or otherwise, or by placing a closed funnel or glass over the ear, as all of these produce the same result by excluding extraneous sounds.

Place the watch or tuning fork against the mastoid in a quiet room, and then obstruct the meatus with the finger. Then try it in a noisy room, and I am sure it will demonstrate that whatever increase we get will bear a direct relation to the amount of extraneous noise that we exclude. That the increase in intensity in this test varies inversely with the amount of extraneous sounds can be easily demonstrated. Place a vibrating tuning fork in contact with the mastoid; then obstruct the external auditory canal. Note the increase. Then again place the vibrating tuning fork against the mastoid and hold a loud-ticking watch one inch from that ear. There is a considerable increase in the sound of the fork if we now obstruct the external auditory meatus with the finger.

In this case there is no increase due to the lateralizing and summation, as there is when we place the fork in contact with the teeth or forehead. The moment we place the fork or watch against the mastoid, we have the lateralization and summation induced at once, as according to my law the sounds pass at once to the hearing center on that side where the intensity is the greater.

The reason we do not get as much increase in intensity when the watch or tuning fork is in contact with the mastoid as when it is in contact with teeth or forehead when we obstruct the external meatus, is that when the watch or fork is placed in contact with the mastoid, we get a lateralization at once, so that when we obstruct the external canal we do not get that increase that is due to the summation.

Then in a quiet room the extraneous sounds are practically drowned out by the intensity of the sound from the watch or fork, which is quite close to the internal ear, similarly as we would be unable to hear a whisper in a room where there is considerable noise. In a room where there is considerable noise, obstructing the external auditory canal with the finger does cause an increase in sound when the watch or fork is in contact with the mastoid, but not as great an increase as we would get if the fork or watch had been in contact with the forehead or

teeth under similar conditions. The reason, as I have stated before, is that when we obstruct the ear with the sound producers against the mastoid we do not get the summation as we get the lateralization and subsequent summation at once when we apply the watch or fork to the mastoid. Whereas, when the fork is on the forehead and then we obstruct the meatus, we get the increase due to two causes, in cutting off extraneous sounds and summation, together with the fact that the sound is not heard as loud when against the forehead as it is when we apply the same to the mastoid, and the sound here being louder will cut off a slight extraneous sound, where the less intense vibrations coming from the watch at forehead would not.

We know that the vibrations from a tuning fork that is placed against the right mastoid reach the cochlea on the left side with sufficient degree of intensity that we would be conscious of hearing them in left ear, if the hearing apparatus on the right side was destroyed, but while the hearing on the right side is present, we get a lateralization and summation at once on that side following the laws of the hearing centers.

That the vibrations reach the cochlea on the left side when the watch or fork is held in contact with the right mastoid is demonstrated in the following test. This test also further proves that the increase in intensity when the external auditory canal is obstructed is due to shutting off extraneous sounds. It also proves that increasing the amount of extraneous sounds will cause a lateralizing to the opposite ear, just as obstructing the external auditory canal by lessening the amount of outside noises to that ear will cause the sound to be lateralized to the obstructed ear.

Obstruct the external auditory canal of the left ear with an electric noise apparatus. (Muffle the electric noise apparatus. We do not need the excess noise that it produces). Place a vibrating tuning fork (256 C-1) on left mastoid, first starting the noise apparatus. We are conscious of hearing the tuning fork in the right ear, and the intensity is increased if we now obstruct the external auditory meatus on that side. Here we have two sounds, the source of each is nearest the left ear.

We hear the louder of these two sounds in the left ear, and not in the right. We hear the other sound in the opposite ear, and we are not conscious of hearing it in the left ear that is nearest the source. That the vibrations from the noise apparatus reaches the cochlea of the right ear we will all admit, but

we are not conscious of hearing that sound in the right ear, as the sound reaches the left ear with greater intensity than it does the right ear. Following the law of the hearing centers, we are only conscious of hearing this sound on that side where the intensity is the greater, as the impulse that reaches each cochlea passes then to the hearing center on that side which receives the vibrations of greater intensity.

Why do we hear the tuning fork in the right ear and not in the left?

The sound of the noise apparatus in the left ear drowns out the vibrations of the tuning fork on that side, similarly as we are unable to hear a whisper in a noisy room that we can easily hear in a quiet room. Therefore, we cannot hear the tuning fork in the left ear. The vibrations of the tuning fork that reach the cochlea of the right ear is what we hear in that ear as they pass to the hearing center on that side, plus perhaps vibrations that reach the cochlea on the left side, but the fact that we hear the vibration of greater intensity in the right ear causes the sound of the tuning fork to be lateralized on that side.

The reason that the intensity is not as great in the left ear as it is in the right ear, even though the source of the sound from the tuning fork is nearer the left ear, is but an example of every-day life, that we all experience where we are unable to hear a watch tick, or a whisper, or even a loud voice. Although we know that we are within the hearing distance of these sounds, yet they are entirely lost to us due to the intensity of the other sounds.

The appreciation of the intensity of a sound varies inversely with the intensity of other sounds of greater intensity. In fact the perception of the weaker sound will be lost entirely to that ear if the difference between the two sounds reaches a certain limit.

If we have 100 candles in a room and one more is added, the extra candle would be just perceptible to the eye. But if a room were lighted with 1000 candles, no appreciable difference would result from the extra candle. In each case a difference of 1/100th of the original strength of the stimulus is needful to cause just an appreciable difference in the sensation, and this is in accordance with Weber's Law.

For light, the increase must be 1/100th of the original strength of the light to cause just an appreciable difference in the sensation.

For hearing, the noise or sound must be increased 1/3 of the original intensity of the sound or noise to cause just an appreciable difference in the sensation.

With Weber's Law and these facts as a basis we can easily understand that when the vibrating 256 C-1 tuning fork is against the left mastoid, and the muffled noise apparatus against the left ear, that the vibrations from the tuning fork that reach the left cochlea are not of sufficient intensity to effect an impulse in the cochlea that will reach the hearing center on that side and be registered there.

The conditions in the right cochlea and right hearing center are different. All the impulses from the noise apparatus reach both cochleas, but according to the law of the hearing centers they are only registered in the left hearing center.

The right hearing center receiving no impulses due to the noise apparatus, readily receives and registers the impulses set up in the right cochlea, and we are therefore conscious of hearing the tuning fork on the right side. Shut off the noise apparatus at the left ear while the fork is in contact with the left mastoid and the sound of the tuning fork will pass from the right ear over to the left ear.

When a vibrating fork is held against the teeth of an individual with normal hearing and the right ear is obstructed with the finger in the external auditory canal, the intensity of the sound is increased and is also lateralized to the obstructed ear. We will all admit that the vibrations reach each cochlea. What happens to the vibrations that reached the cochlea on the left side?

Instead of passing to the hearing center on the left, they pass to the hearing center on the right via nerve tracts. This is according to my law of the hearing centers and is the cause of the summation.

That the increased sound in an obstructed ear, that is, when we obstruct the canal with the finger, is not due to increased resonance is shown by the fact that we get an equal increase when we place a large funnel over the ear or a receptacle with a capacity of several gallons over the external ear; that the increase in sound is not due to preventing the passage of undulations outward from the ear when the vibrating body is brought in contact with the cranial bones, for with the ossicles and the membrana absent there is even less obstruction to outgoing vibrations than when they are present, but even then we get

the same increase in sound as when the external canal of a normal ear is obstructed with the finger. In fact Jankan's test in obstructive deafness external to the stapes the tympanic structures being in a state of increased tension will allow of rather more vibrations to pass out.

Steindruegge considers that the absolute or relative increase in bone induction in obstructed deafness is due to hyperesthesia of the auditory nerve, while this condition may by chance be present in an isolated case, it is certainly not the cause of the increased bone conduction, as the vast majority of cases that lateralize the tuning fork do not present any evidence of auditory hypersensitiveness.

In cases where the membrana tympani has been destroyed, pressing the stapes inward with a probe, is said to prolong bone conduction, which Siebenmann says is due to increasing the labyrinth pressure. I think that it is more likely to be due to lessening the extraneous sound from without that is the cause of the increase in sound in such a case.

Schwabach first found that in obstruction of the sound conducting apparatus the vibrating tuning fork is heard longer than under normal conditions. One reason why an ear with obstructive deafness hears the tuning fork longer when applied to the mastoid on that side than does the examiner with a normal ear is that extraneous sounds in the examiner's ear will drown out the tuning fork earlier in the test than the patient who hears little or none of them, depending on what degree of hearing that he has retained.

S. Bard claims that the bone conduction of the vibrations of a tuning fork is more a matter of sensibility of bone; that the acoustic factor predominates at first, but as the tuning fork vibrations grow less intense, the tactile perception predominates and persists after the acoustic has died out. If this be a fact then we will have to change slightly the wording of Schwabach test as follows:

In obstructions of the sound conducting apparatus the vibrating tuning fork, when brought into contact with the cranial bones, its acoustic properties are heard longer than under normal conditions. As we hear it louder in obstructive deafness we must hear it longer. How to determine the time that tactile perception predominates I will leave for its author.

To sum up all the facts which we are able to present we are able to prove but two factors in the lateralizing and increasing

the sound in obstructive deafness; i. e., Cutting off extraneous sounds and the summation in that hearing center.

1. Gradenigo test proves what we all know, that obstructive deafness renders the transmission of vibrating impulses more difficult in proportion to the degree of the obstruction.

2. Jankan's test shows that in obstructive deafness where the obstruction is external to the stapes that a stronger tone will come from the obstructive ear than from the normal ear. This shows that instead of an interference with the egress of sound received from the labyrinth or in that region, that the egress is greater than in the normal ear.

3. That obstructing the external meatus with finger when the watch or fork is in contact with mastoid on that side causes little or no increase in intensity of those sounds, in a fairly quiet room, while it does in a rather noisy room. This shows that shutting off extraneous sounds is the chief factor in the increased intensity and the lateralizing.

4. Obstructing one ear and increasing the extraneous sounds going to that ear lateralizes the sound in the opposite ear and when the extraneous sounds are then removed but the ear still obstructed with the noise apparatus the sound of the tuning fork passes over to the obstructed ear.

5. That increasing the sound in one of two normal ears with a C-1 256 tuning fork while the ticking watch is held against the teeth increases its intensity, which is still further increased by obstructing with the finger the opposite ear.

6. That two sounds which are equal in all their properties except intensity, and one of which goes to each ear (all other sounds being excluded), we are only conscious of hearing them in the ear on the side where the intensity is the greater, and they are heard with greater intensity than if the sound of lesser intensity was cut off from the other ear. This increase in due to summation alone.

7. That the reason that a sound from a tuning fork is lateralized in an obstructed ear and we are not conscious of hearing the sound in the opposite ear, although we know that the vibrations do reach that ear is because it follows the law of the hearing centers which I have previously quoted.

8. We get the same result when we obstruct the external auditory canal with the finger as when the tympanic membrane and ossicles are absent when the vibrating fork is placed in contact with teeth or forehead.

9. As to the amount of the vibrations that escape through the external canal or in the region of the internal ear or limiting the egress of the vibrations by the obstructions in the middle ear, it cannot account for the increase in intensity that we get when we obstruct the ear, as we get the same in increase if we place a large receptacle which fits closely over the auricle, as we do when we obstruct it with the finger.

I must admit that it is the cause of a very slight increase, but the increase is so slight that we cannot demonstrate it.

If we place a funnel 3½ inches in diameter over the occiput when a C-1 256 vibrating fork is in contact with the teeth, the sound of the fork can be heard through the tube at 15 feet. This shows that the vibrations pass out of the skull at all points.

10. That the blocking of the external auditory meatus or obstructive deafness, and thus interfering with the egress of the sound in this region, has little or nothing to do with the increase in intensity of sound in these conditions, is shown when a watch or tuning fork is placed in contact with the mastoid. In a quiet room I cannot myself note any appreciable increase, whereas, in a noisy room there is.

The increased resonance when we obstruct the external auditory canal and the reflection of the sound waves transferred through the cranial bones to the air in the external auditory canal, and thence to the membrana tympani and ossicles does not influence the intensity to any extent, not even enough so that it is appreciable.

The summation of stimuli is best demonstrated with a tuning fork and a rubber tube with a ½-inch hole and 3/32-inch wall, and about 2½ feet long.

Place ends of tube opposite each ear. Place vibrating forks about three or four inches from right ear and after it is vibrating while in contact with tube for about ten seconds remove end of tube that is near the left ear and the sound which is only heard in right ear loses considerable of its intensity. This may be increased by bringing the end of tube close to left ear again.

Politzer (I 173) first showed that when the finger is inserted into the normal ear in some cases of unilateral affections of the sound conducting apparatus, the increased sound perception of the vibrating tuning fork will be transferred from the affected ear to the normal ear, and upon removing the finger the sound will again return to the affected side. The only reason for the lateralizing of the sound is that when the finger was placed in the

normal ear, it shut out more of the extraneous sounds than were shut off by the obstructive process in the affected ear and the vibration of greater intensity reaches that ear the sound is lateralized according to my law. The vibrations of the tuning fork that reached each cochlea were of equal intensity, but we know that other or extraneous sounds will lower the power of perception of any sound depending on the intensity of such extraneous sounds which may be so intense that perception of the sound of the test cannot be heard at all.

Stenger's test showed that the perception of a tone in one ear can preponderate and completely mask the perception of tone in the other ear. He used tuning forks, the tones of which were exactly alike and noted the distance at which one is heard in the normal ear. The second fork is now brought nearer the supposed deaf ear. In case of true unilateral deafness the hearing distance for the tuning fork on the normal side remains unchanged. If, however, there is unilateral, simulated deafness, the normal ear perceives the tone only when the tuning fork is brought nearer the ear than the fork on the apparent deaf ear.

My test on malingering is developed from the same principle, but I use a single tuning fork and a single rubber tube going to each ear. My voice test is also along similar lines.

At present I am trying to demonstrate from a physiological point of view how these phenomenon take place and the laws that govern them.

*In my test for malingering with the voice, it beautifully demonstrates how we lateralize the more intense of two sounds in one hearing center.

With a rubber tube with 3/8 inch hole and 3/32 inch wall, the normal ear can hear a whisper at about thirty-five (35) feet.

Take two tubes each ten (10) feet long. Connect them with a funnel which is placed over each ear. Both tubes are connected with a mouth-piece which consists of an ether cone covered by tin at the smaller end, and from which two tubular extensions enter the cone. With the two tubes of equal length the patient is conscious of hearing the whispered voice in both ears, or he may tell you that he hears one voice in the center of his head.

Make the tube going to the right ear one, two, or three inches shorter than the one going to the left ear. Shortening the rubber tube is accomplished by slipping it further on the

*Boston Medical & Surgical Journal, Aug. 15, Sept. 26, 1918.

tubular extensions of the mouth-piece or the funnel. Start counting (whisper) into the mouth-piece, say from one to twenty-five. When we have whispered five or six numbers, disconnect the tube going to the left ear, which is the longest tube, remembering that the patient is only conscious of hearing the numbers counted in the right ear, which is connected with the shorter tube. What happens when the longer tube is disconnected from the left ear? The patient will tell you that he does not hear the count as loud as he did when the both ears were connected. We keep right on counting, the tube having been again connected with the left ear. What happens now? One or two things may happen.

First, the patient may not hear anything in the left ear, and if he does not, he will tell you that he hears the whisper louder in the right ear.

Or the patient may hear the whisper in both ears, but the sound will only remain in the left ear, while three or four numbers are being counted, when suddenly he will have a sensation of the sound leaving the left ear. It then becomes lateralized in the right ear, and he also tells us that he hears the whisper louder than at the time he heard it in both ears.

This test demonstrates that when we are conscious of hearing a sound only in one ear, when both ears are connected with the source of the sound, that the vibrations do not reach the hearing center on the side that receives the sound of lesser intensity. They evidently pass directly from the cochlea on that side by nerve tracts to the hearing center on the opposite side.

The sound in the right ear is not increased in the same proportions as to the amount of the sound that passes from the left ear by the tracts to the right hearing center. The increase in sound in the right hearing center when the impulse of greater intensity reaches that side, plus the intensity of the weaker impulse that reaches the left ear and passes to the right hearing center follows Weber's Law.

For how surely the hearing centers lateralize two similar sounds of unequal intensity is demonstrated in following test:

Two ten (10) ft. tubes connected as in previous test.

Whisper into mouth-piece and patient will tell you that he hears the whisper in both ears. Shorten the tube going to the right ear and he will hear the whisper only in the right ear. Some individuals lateralize easier than others. In some cases we must shorten the tube 3 or 4 inches and even more.

But it shows the power that we have of lateralizing, for in the test we use tubes ten (10) feet long; the patient can hear a whisper through at thirty-five (35) feet in either ear, and yet by making the ten (10) foot tube one or two inches shorter going to one ear, the patient is unable to hear the whisper in the ten (10) foot tube and will only hear it in the tube that is nine feet eleven inches.

In my voice test to detect malingering I have changed the technique so that we may better determine the extent of the hearing in the supposedly deaf ear. Find the distance that he can hear in his good ear through a rubber tube. Attach the same length of tube to funnel at bad ear. We may place a control on test by having another tubular extension attached to mouth-piece. Attach rubber tube to this, that is the same length that passes to the malingerer's good ear. This goes to a second party whose hearing is normal.

It is always best to have a tube going to the supposed malingerer's good ear that is two or three feet or even five feet inside the hearing distance of that ear, as we must be sure that he is capable of hearing the whisper in that ear at all times. We now whisper through three tubes. Our patient and our control, of course will admit hearing, as of course, the patient hears the whisper in his good ear.

We now start to shorten the tube that goes to the patient's supposedly deaf ear and we keep testing and continue to shorten that tube until we reach a point where the patient, if he is a malingerer, will hear the whisper only in his supposedly deaf ear, when of course, he will deny that he hears it. When the malingerer changes his answer from "I hear" to "I do not hear," we know then that he is hearing in his supposedly deaf ear, and that we are surely dealing with a malingerer. This will then be the measure of the hearing that he has in his deaf ear.

When we reach that point on the tube going to supposedly deaf ear that patient claims he cannot hear the whisper, we disconnect with his bad ear and whisper into the tube that is now only connected with his good ear and our control, and of course, the malingerer will admit hearing again. We can repeat this procedure two or three times.

Our control always hears the whisper and the malingerer should always hear it, as we always have a tube connected with his good ear that is inside the limit of hearing distance of that ear. As soon as he hears louder in his supposedly deaf

ear, he will deny hearing at all, as he then does not hear it in his good ear. If he should admit hearing when he was hearing the whisper in the supposedly deaf ear, we disconnect the tube going to his good ear and in that case he would admit himself that he was caught.

LAW OF HEARING CENTRES.

When two sounds which have similar properties except intensity, are transmitted one to each other, both impulses will pass to the hearing center on that side where the intensity is the greater, and we are only conscious of hearing the sound in that ear. If now, two more sounds are transmitted, but the one of greater intensity reaches the ear on the opposite side, we will be conscious of hearing the second sound in that ear. Both sounds being simultaneously registered and heard only on the side where the intensity is the greater.

When two sounds which have similar properties except intensity, are transmitted one to each ear, both impulses will pass to the hearing center on that side where the intensity is the greater, and there will be a summation of the two impulses, and we are therefore conscious of hearing the sound louder than if the sound of lesser intensity did not reach the ear, and also if both sounds reach each ear with equal degrees of intensity. The increase due to the summation is not equal to an arithmetical addition but follows Weber's Law.

If a C-2 512 and a C-1 256 vibrating tuning fork are placed one inch from right ear, we can detect the tone due to each fork. We can also hear them at any distance within the hearing limit. But if we place the C-2 512 fork against a rubber tube two feet long, each end of which goes to each ear we will only be conscious of hearing in the ear that is nearest to the fork, although the ear that receives the vibrations of lesser intensity is within the hearing distance of the fork.

If we now place a C-1 256 fork on same tube and four inches from the ear that receives the vibrations of lesser intensity from the C-2 512 fork we will be conscious of hearing the C-1 256 fork in that ear, and the 512 fork in the other ear. In this test the C-2 512 fork was within the hearing limits of both ears, but we were only conscious of hearing that fork in the right ear, which was nearest.

That the vibrations that passed to the left ear did not pass to the left hearing center but crossed over to the right hearing center is proven by the fact that when we placed the C-1 256

fork four inches from the left ear, we were conscious of hearing that fork in the left. We are only conscious of hearing a sound in the ear that is on the side of the hearing center that receives the stimulus of greater intensity and two sounds that reach each ear, with different degrees of intensity (if the pitch and timbre are the same), will always pass to the hearing center that is on the side that receives the vibrations of greater intensity, it being understood that the ear tracts and hearing centers are normal.

That these sounds of lesser intensity do not reach the hearing centers when we have the C-2 512 and the C-1 256 forks on the rubber tube is proven by the fact that when these two forks are sounded together on the rubber tube when the rubber tube is only connected with one ear the trained ear can detect the tone due to each fork, and is conscious of hearing it in the ear that is connected with the rubber tube.

TEST TO SHOW HOW THE TWO HEARING CENTRES ACT AT THE SAME TIME.

Previous to the test two records have been made by one person on a dictograph. Into each record is read different sentences. Care should be taken in the selection of the disks, so that the pitch and timbre may be similar when they are produced on the dictaphone. The intensity can be regulated on the dictaphone. All three characteristics of sound in the sound from the dictaphone should be as near alike in the two records as possible.

Connected to the dictaphone is a rubber tube which is connected by a T to two short rubber tubes that are inserted into each meatus, when the machine is in use.

Two funnels each with two outlets are placed one opposite each ear. Two tubes ten (10) feet long are connected to dictaphone No. 1 at the ear pieces. One of the ten (10) foot tubes is attached to one of the outlets on the funnel at the right ear. The other tube is attached to one of the outlets of the funnel at the left ear.

Dictaphone No. 2 is likewise attached by two ten (10) foot tubes, one going to the second outlet of funnel over right ear and the other ten (10) foot tube going to the second outlet of funnel which is placed over the left ear.

Both dictaphones are started. What happened? If each ear were connected with a single dictaphone we can easily hear what the dictagraph has recorded on the record.

In this case we have the right ear connected with both dictaphones, and the left ear likewise connected. The spoken words from dictaphones No. One and No. Two pass to the right hearing center and also to the left hearing center. We cannot understand a word that is sent by the two dictaphones. Nothing but a babble of unintelligible noises like one might hear from a gallery while several people were talking all together on the floor below.

We shorten the tube that passes from dictaphone No. 1 to right ear about three inches. Following my law of the hearing centers we get the following result: The tube passing from dictaphone No. 1 to right ear is now shorter than the tube going from dictaphone No. 1 to left ear; therefore, we will hear dictaphone No. 1 loudest in the right ear and the sound from the tube going to the right ear will pass to the right hearing center as will also the sound from the tube that passes through the tube from dictaphone No. 1 to left ear, and accordingly we are only conscious of hearing the sound on the side that receives the sound of greater intensity. We are at once conscious of hearing the voice sounds from dictaphone No. 1 in right ear.

Now that the sounds from dictaphone No. 1 are heard plainly in right ear they must pass only to the right hearing center. The left hearing center not receiving any of the sound impulses from dictaphone No. 1, is only connected with dictaphone No. 2, the sound of which passes to that the left hearing center. We therefore hear the sound from dictaphone No. 1 in the right ear and from dictaphone No. 2 in the left ear; although we know that the sound from No. 1 dictaphone passes to both ears, as does the sound from dictaphone No. 2.

A further proof that the sound passes from dictaphone No. 2 to left hearing center via the right ear is demonstrated by disconnecting the tube that passes from dictaphone No. 2 to right ear. We do not hear the sound as loud in the left ear as when both tubes are connected with dictaphone No. 2 and both ears.

Similarly, if we disconnect the tube that passes from dictaphone No. 1 to left ear, we do not hear the sound as loud in the right ear.

The increase in the sound if we again connect the tube and the fact that the increase is noted in the ear on the opposite side from where the tube is attached is sufficient to show that

it passes from one ear over to the hearing center on the opposite side, there to be united with the sound that reaches that hearing center from the ear on the same side.

In the first part of the test I usually have considerable trouble in getting all the four tubes and connections exactly alike so that the conduction of the sounds will be of equal intensity when it reaches the ear from all of them, for if the intensity is greater in any one of the tubes when it reaches the ear, we will get a lateralizing of the sounds.

To cause the sounds from the two dictaphones to be lateralized is especially easy, as it is considerably easier to get all not alike in their conductivity than all exactly alike. The several individuals who tried the above test all could hear, and repeat what we used in the test at twenty (20) feet. In the test I used what they heard in each dictaphone through the same size tube a similar tube in ten (10) foot lengths.

Local Anaesthesia in Otorhinolaryngology. G. LAURENS, *Presse méd.*, Par., 1919, xxvi, 300.

Either the infiltration or regional methods of inducing local anaesthesia may be used and at times both may be combined. The advantages to the surgeon and patient are: simplicity, minimum, toxicity, and, in laryngeal operations, diminution in the shock and asphyxia.

The surgeon must inspire absolute confidence in the patient and assure him that the pain will be reduced to a minimum. The psychic, suggestive, and moral effect must not be overlooked. The technique ought to be above reproach, the anatomical points known exactly, and the instrumentarium correct. Gentleness is essential in dealing with the tissues, and in opening the cranium and sinuses an electric motor should be used instead of the gouge and mallet. The anaesthetic should consist of a 2 per cent novocaine solution with the addition of 25 drops of adrenalin for each 100 cubic centimeters. A larger amount of adrenalin makes possible the occurrence of postoperative haemorrhage, haematomata, and abscesses.

To anaesthetize laryngeal cavities in the course of thyrotomy and laryngectomy the author recommends injecting a solution of cocaine through the cricothyroid membrane.

ACUTE MASTOIDITIS; A CLINICAL STUDY BASED ON CASES SEEN IN THE OTO-LARYNGOLOGICAL SERVICE, U. S. GENERAL HOSPITAL 14, FORT OGLETHORPE, GEORGIA.

DR. THOMAS J. HARRIS, Lt. Colonel, M. R. C., New York.

Acute affections of the middle ear were a frequent occurrence at all the army camps during the year of 1919. Several excellent reports have been made regarding them, including one by Major Lathrop at Camp Shelby, Miss., and another by Major Scott and others at Ft. Riley. There can be no question that they are correct in ascribing them to the close association of large bodies of men under unusual conditions and to the virulence of the infection. The camps situated in Chickamauga Park, Georgia, were no exception to this. It is probably true that no great differences in the clinical picture existed in the cases coming from them to U. S. General Hospital No. 14, Fort Oglethorpe, Ga. Certain features, however, of a pathologic and diagnostic nature were of such interest to us and so unlike what we had previously met with that we desire briefly to report them at this time.

Two diseases stood out above others as the producing cause of these acute middle ear infections, namely, measles and influenza. We propose to confine our remarks in this paper to the ear complications of these two diseases.

MEASLES.

Measles proved to be one of the most serious maladies that the army doctor was called upon to meet. Contrary to the general impression in civil life, it proved to be a disease apt to be followed by grave complications of which pneumonia and otitis media were the chief.

During the winter and spring the medical wards of the hospital were continually filled with cases of bronchitis or bronchial pneumonia, mumps, and especially measles. Of the cases of measles, a majority was complicated by acute laryngitis and rhinitis. Of these, the number was large who in the course of the disease developed an acute affection of the middle ear. Many of these, in spite of the most prompt and energetic treatment, went on to acute mastoiditis, requiring immediate operation to avoid serious or fatal intracranial complications. The number of ear cases treated from January 1st to July 14th was 1,685, of which 607 were cases of measles. The symptomology

of the cases of otitis media acuta occurring in measles was strikingly characteristic and differed entirely from the ordinary classical picture met with in civil life. The patient, more or less toxic, with elevation of temperature, pronounced leucocytosis and racked with cough from his bronchitis or bronchial pneumonia, would be found lying entirely free from all pain, either subjective or objective as far as the ear is concerned.

In fact, it was our experience that in a great majority of these cases, the ear complication was only discovered in the course of a routine examination. At the most, the patient would admit upon direct questioning that there was a feeling of stuffiness in the ear. The examination of the ear would show no characteristic picture. Usually the landmarks were obliterated and there was a boggy, unhealthy appearance of the drum membrane. If seen extremely early in the infection, there might be a slight injection in the handle of the malleus and Shrapnell's membrane. At a variable time after the onset, bulging of the drum took place in the posterior upper quadrant. To wait for this to occur almost certainly meant mastoiditis and operation. It was our rule freely to open the drum immediately upon the discovery of any change in its appearance. Often the incision was followed by muco-purulent discharge. If the incision had been made early enough, if the infection was not too virulent and if unceasing vigil had been exercised in maintaining free drainage by repeated paracenteses, the case gradually went on to convalescence and recovery. If, on the other hand, free drainage had not been established or maintained, if the organism present, usually a streptococcus or pneumococcus, was exceedingly virulent, if there was a diseased condition of the nose, and finally if the resisting powers of the patient had been too much lowered, the inevitable course of the disease was toward an extension of the process from the attic to the antrum and the mastoid cells. Acute mastoiditis appearing in a patient suffering from measles was, as a rule, just as free from pain as was otitis media. It is almost impossible to believe that a patient could have a mastoid full of pus and yet insist that he is not suffering and scarcely admit any tenderness on pressure either over his antrum or over his tip. The explanation for this is not altogether easy. In some cases it was undoubtedly due to a thick outer cortex. In many, however, we are inclined to feel that it lay in the general marked torpor of the patient. The only signs that could be depended upon were changes in the upper posterior wall of the canal. While

it was the rule to find these to a greater or less degree, according to the duration of the disease, we met cases where they were almost, if not altogther, lacking. The discharge was wont to be profuse and frequently under pressure as shown by pronounced bulging in the line of incision.

In several cases an extensive destruction of the drum membrane took place which at first we were inclined to ascribe to the local anesthesia used. We were finally forced to the conclusion that it was due to the virulence of the organism present. Upon the recognition of changes in the auditory canal wall, immediate operation was indicated. In rare cases, even where these changes were absent, with excessive discharge or destruction of the drum membrane continuing for a number of days, it was our rule to operate. Any definite day of the disease for operation was out of the question. We never operated too early, even if it was only the fourth or fifth or sixth day. Often we should have operated earlier. Operation was wont to disclose extensive changes in the mastoid cells. If the operation had been performed early enough, the progress to recovery, while usually unduly slow, was uneventful.

In the after treatment, we tried in a number of cases Dichlomentine T. While in some cases it proved satisfactory, we could not satisfy ourselves that it was materially better than gauze packing properly introduced. In one particular our experience was especially gratifying. Without exception the cases operated upon were exceedingly sick men. In all of them the employment of the usual anesthesia, ether or chloroform, could not have failed to produce pronounced shock and thus serve as a deciding factor against the patient's recovery. In place both of ether and chloroform it was our rule to use nitrous oxide gas and oxygen. The immediate and later results were truly remarkable.

The records which were kindly prepared for me by the chief anesthetist of the hospital show that from April 3 to July 26, 31 cases of mastoiditis complicated by measles, were operated under nitrous oxide gas and oxygen. Of these, 10 had bronchial pneumonia at the time of the operation. The shortest time for administration was 40 minutes and the longest was 3 hours and 45 minutes. The average time for administration was 1 hour and 15 minutes. All patients were conscious before the dressing was completed. One case requiring an anesthesia of nearly 4 hours was particularly noteworthy. It was a man suffering from measles, double bronchial pneumonia and double

mastoiditis. Both mastoids were found diseased and requiring operation. There was absolutely no shock following the operation and the patient's temperature the next morning was virtually normal. This was true of all the cases where this form of anesthesia was employed. Beyond question it is the ideal anesthesia for such cases and commends itself strongly for all extensive or serious operations.

Two important deductions forced themselves upon us as the result of our observations of otologic complications of measles in army life:

First, that they are so unusual in their symptomatology that the only certain means of recognition is the examination of the ear itself. This, in a civil hospital service, is the province of the specialist. In army life dealing with large bodies of men such special attention manifestly was often impossible. This necessitated that every ward surgeon should be capable of making a satisfactory otoscopic examination. So important did this seem to us that in connection with the work in the School of Oto-laryngology instruction was given to every member of the School of Surgery in order that he might acquaint himself with the normal and thus be able to recognize an abnormal appearance of the membrani tympani.

Second, far outweighing all measures addressed to relieving infection of the ear is prophylaxis to prevent such infections from developing. Much was done in this direction both at Oglethorpe and in other cantonments throughout the country. All improved sanitary procedures, it is unnecessary to say, are to be carried out. In addition to these, local treatment of the upper respiratory tract in some form or other is distinctly indicated. What the best form of this has not been definitely determined. Gargles, such as a warm saline solution or Dobell's solution recommend themselves. For a time we used in atomizer form a 2 per cent. solution of Dichloramine T which, properly prepared, is fairly free from irritation. The suggestion made by Lt. Colonel Harris P. Mosher, of the erection of steam huts for this purpose such as are now used in the British army, particularly recommends itself. Any or all means such as these which will cut down the inroads of the bacillus are the only sure means to complete success in this important line of work.

INFLUENZA.

Contrary to general expectation and probably to the common impression today, otitis media was not a frequent complication of influenza. Out of a series of some 7,000 cases, only 120 cases of

acute middle ear suppuration were seen at General Hospital No. 14. There were 1,600 cases of pneumonia in this number and 66 of the cases of otitis media occurred among these. This would give a percentage of 1.75, or a little over 4 per cent. in the pneumonia cases. While the number was not very large, it afforded sufficient opportunity carefully to observe and note any special features. In several particulars, these were of sufficient interest to deserve brief mention.

We desire particularly to refer to

1. The drum picture.
2. The changes in the posterior superior canal wall and their significance.
3. The value of thickening of the periosteum as a diagnostic sign of mastoiditis.
4. The value of the roentgengram as an aid in diagnosis.
5. The operative findings.
6. The time fr healing.

The onset of otitis media was apt to be insidious as in the cases of measles. Whether complicated by pneumonia or not, the cases of influenza were all suffering from elevation of temperature from 101 to 104. In some of the cases, there was severe pain when the ear was infected; in others little or no pain was ever complained of. Indeed pain and temperature as a diagnostic symptom had little value. The chief reliance in diagnosis had to be placed upon the appearance of the drum membrane. This was so characteristic that it should be referred to at some length. It has been admirably described by my former assistant, Lt. F. T. Hill,* in a paper on the subject, as follows:

"Otoscopic examination of the case within two or three hours after the onset of the pain would show distinctive changes in the membrana tympani, always of a vesicular type. There was a marked redness and some bulging of Shrapnell's membrane in almost every case. There was frequently a large hemorrhagic bleb bulging outward even for two or three mms. The short process often could be seen. This was the typical case seen within two or three hours after onset.

The cases seen later showed more swelling, especially of the superior posterior portion, diffuse reddening of the membrana tympani and loss of all normal land marks. The distinctive feature was a bleb formation. Often there were two or three of these always superior, either anteriorly or posteriorly and

*The Laryngoscope, June, 1919.

often dissected marginward beyond the annulus and to the wall of the external auditory meatus. These were of considerable size, often being 5 or 6 mms. in diameter, obscuring a good view of the membrana tympani. The eccentric layer of the squamous cell epithelium making up the stratum cutaneum of the membrana tympani readily explains this traveling of the blebs laterally to the canal wall.

There was no tenderness over the mastoid process at this stage. Incision of the blebs evacuated a small amount of bloody serum. In the older cases this could be expressed from the vesicle only with some effort, as if some clotting or organization was taking place. These blebs were true vesicles beneath the stratum cutaneum of the membrana tympani. After evacuation of these, the drum appeared red and swollen with the marked involvement of Shrapnell's membrane previously mentioned, the degree or extent depending upon the time involved. Incision of the membrana tympani in the earlier cases gave relief to increased pressure within the middle ear, followed by considerable bleeding. Later this became a profuse sero-sanguinous discharge. In the cases seen at a more advanced stage, incision evacuated this sero-sanguinous discharge immediately. The pain generally subsided about two hours after the incision of the membrana tympani. The sero-sanguinous discharge continued rather profusely for several days, then gradually changed in character to a thin purulent discharge, which later became of thicker consistency, apparently due to secondary infection.

Presumably we had a very acute suppurative otitis media, rapid in onset of naso-pharyngeal origin. Almost from the onset, a marked epitympanic involvement became noted. An outstanding feature was the early obliteration of the superior angle of the external auditory canal and membrana tympani, sometimes seen on the second day of the disease. With this there was increased swelling of the membrana tympani itself and often further bleb or vesicle formation, though after incision of the drum no new extra marginal blebs were seen. With the obliteration of the superior angle, Shrapnell's membrane became pushed downward. The line of incision frequently was depressed inwards by the increased thickness or swelling of the membrana tympani. Often a bleb or vesicle would protrude out of the incision.

"Generally there was what seemed to be a hyperplasia of the mucous membrane of the middle ear. Sometimes this protrud-

ing through the opening of the membrana tympani would give the appearance of granulation tissue except that it did not bleed so easily. In one case this hyperplastic tissue coming from the membrana tympani higher up dissected away the superior angle of the drum, giving the appearance of a polypoid mass. This was freely movable without discomfort to the patient and did not bleed." We do not desire to give the impression that it was possible to note in all cases these progressive changes. In some cases the pathologic process was well advanced when first seen.

We would emphasize that almost without exception the subject of this ear complication was already a very sick man. The course of the disease varied. In many cases after free and often repeated incision of the drum, the discharge gradually subsided and resolution took place. In others, 21 out of 120, changes took place in the mastoid process itself, demanding operation. The decision when to operate on these cases was not an easy one. As we have just mentioned, the patient was already running a high temperature. No help could be gained from the temperature chart. This also ruled out making use of the blood picture as far as the blood count was concerned.

Often careful and repeated chest examination left us in doubt as to the development of pneumonia and how to account for the continued elevation of temperature. Mastoid tenderness and especially pain was an unusual symptom. There was apt to be profuse discharge, as has been described. Changes in the posterior superior canal wall were wont to be met with. While in the majority of the cases the operative findings showed that these changes were dependent (as has been generally believed) upon the disease of the mastoid cells bordering the canal, in several such cases changes could not be demonstrated at the time of the operation. This led us to regard with less confidence such changes as positive indication in itself for operation and to believe that as a result of numerous canuliculi found in the inner portion of the posterior osseous external canal, there existed actually only an oedema of the canal wall due to an extension laterally of the epitympanic process seen first as an obliteration of the upper angle. The membraneous canal is less firmly attached to the osseous in this plane.

More reliable was found to be the *thickening* of the periostium over the mastoid. In the case of unilateral mastoiditis it was often possible at an early stage in the disease to detect by careful palpation a velvety thickening on the affected as compared with the healthy side.

Finally, we came to put great reliance on the value of good and often repeated roentgenograms as an aid in diagnosis. In this we heartily agree with Scott and others in the estimate they place upon them. We cannot follow them, however, to the extent that there exists a distinct type of cell shown by the x-ray picture which does not call for operation. The x-ray is a valuable aid to diagnosis but it has its limitations. A good picture is essential and is not easy in a bed patient to obtain with a portable apparatus. A poor picture can easily lead to error. Expert interpretation is required. A previous mastoiditis causing sclerosis seriously interferes with arriving at a correct conclusion. Simultaneous involvement of both mastoids prevents comparison, which is all-important.

The *operative findings* varied materially according to the time of intervention and virulence of the infection and type of mastoid. In some cases where the mastoid was opened early in the disease, little or no changes were noted. These cases went on to uncomplicated recovery, the temperature, as a rule, dropping to normal soon after operation. In cases free from previous mastoid disease, it was not uncommon to find extensive cell involvement both in the zygoma, post-sinus region and the tip. Repeatedly pus was found in the second layer of cells adjacent to the posterior canal wall.

In our experience there was no uniformity in the nature of the organism recovered and contrary to the findings at Camp Shelby and Fort Riley there was no prevailing organism recovered with sufficient frequency to warrant the term "epidemic mastoiditis."

One of the most interesting features in our series of mastoid cases totaling for the year 160 cases out of a total of approximately 3,000 cases of middle ear trouble was the slowness of healing. Instinctively the thought presents itself that these operations had been incomplete. The fact is that extreme care was taken to search out and remove every area of disease. The real explanation is to be found in the poor subject already reduced by disease and in the virulence of the organism. Even under such conditions attention to securing an early closure of the wound by primary partial suture and care in the introduction of the gauze packing—not too much nor too long-employed, effected very satisfactory results.

TIGHT STRICTURES OF THE ESOPHAGUS IN CHILDREN, DUE TO LYE BURNS.

Dr. George F. Keiper, Lafayette, Indiana.

Permit me at the outset to qualify the title of this paper, to refer to those strictures of the esophagus which the general practitioner or surgeon is unable to dilate with the ordinary instruments at his command, after blind attempts. Because of this failure the case is referred to the laryngologist as a last resort before attempt is made to perform gastrostomy to relieve the patient of water hunger, which is always to be more feared than food hunger. A person may go forty days without food, but he cannot well survive three days of water hunger. In this connection I wish to reiterate all that I wrote in the paper read before the 1912 meeting of this society and refer to the excellent papers of Mosher and Jackson subsequently presented on this subject.

The etiology of strictures of this sort lies primarily in gross carelessness. Too often concentrated lye in solution is left within easy reach of small children, after the kitchen sink has been cleaned. The inquisitive child proceeds to investigate the contents of the vessel containing the corrosive poison and very often drinks it. The usual distressing results follow invariably in a greater or less degree, and sooner or later the unfortunate little one experiences difficulty in attempting to swallow its food or drink. A thimble full of such solution is sufficient to cause stricture as in the case which I will report, and illustrate. These lyes and cleansers are corrosive poisons and if sold by a druggist he must affix the poison label with the skull and cross bones, as well as information as to the antidote to be administered in case of accidental poisoning. But the grocer next door sells them without these precautions and with impunity.

In the barn on the farm is usually found a medicine chest containing the remedies used in treating the live stock. In it is usually found a bottle of hydrochloric acid, which is used to burn warts off the legs of horses and cattle. It is usually within the reach of the children and in two instances in my practice the acid had been ingested by small children with resultant inanition; but for a wonder in neither instance was there a

*Read before the Midwest Section of the American Laryngological, Rhinological and Otological Society, Denver, Col., March 1st, 1919.

stricture formed. However, the mucosa of the stomach was in a large measure destroyed.

Haste in dilating strictures of the esophagus after the ingestion of the corrosive fluid is to be deprecated, for fear of producing perforation of the already softened esophagus, rendered thus by the chemical reaction of the poison upon its tissues. In fact the stricture does not form immediately after the accident. It may take several weeks to form it, before it is noticeable in the inability of the patient to swallow well.

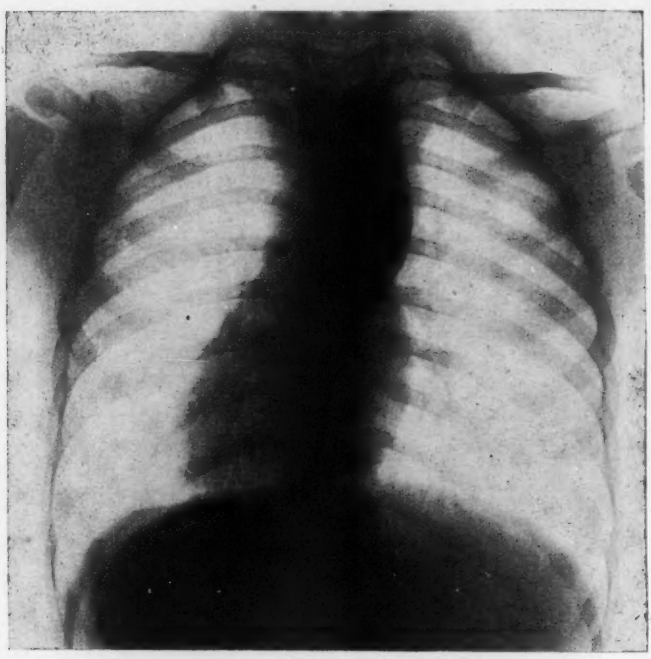
Again, no attempt should ever be made at bouginage until two sets of x-ray plates are made to locate accurately the site, extent and character of the stricture, one laterally and one anterior-posteriorly, and in each instance stereoscopically, after the patient has swallowed the usual bismuth mixture. These are absolutely necessary in order to obtain the information needed. Moreover, no bougie should ever be passed except under direct inspection through the illuminated esophagoscope in position. Blind bouginage is too dangerous a procedure. In thirty-four cases of cicatrical stricture, examined postmortem in the pathologic-anatomic institute in Vienna between the years 1877 and 1886, death was due to perforation by the bougie in no less than fourteen. In fact in the case reported it was impossible to pass the very small bougies without the illuminated esophagoscope in position.

As large an esophagoscope as possible should be passed, gently, in order to dilate the esophagus and thus render as easy as possible the location of the ofttimes the very narrow opening into the stricture. In all manipulations the utmost care and gentleness are to be observed. Above all things we must avoid sandpapering the gullet, to paraphrase a saying of the genito-urinary surgeons. All instruments are to be carefully annointed before introduction.

The great majority of these strictures occur in small children. Shall we give a general anaesthetic each time we introduce our instruments? Personally I believe such frequent anaesthesia to be highly dangerous. The normal esophagus has but very little sensation, and what shall we say of the esophagus damaged in whatever nerves of sensation that it possessed before the accident. We may need to make as many as sixty such dilations before we can dismiss the patient as cured.

The child is pinned in a sheet or blanket in such fashion that it will be unable to use or move hands or feet, in resistance to

the introduction of our instruments. The child is lain flat on the operating table while the assistant holds its head over the end of the table where the operator sits. A nurse holds the body of the child in position on the table. If the little one refuses to open the mouth for the introduction of the mouthgag, tickling the fauces with a fine probe introduced between the teeth will quickly cause the mouth to open. No anaesthetic, even local, is used in my practice. When once the child realizes what is



Skiagram of Esophageal Stricture.

demanded of him or her, it is surprising how docile it becomes, and offers no resistance at all.

These strictures are often multiple and tortuous. For dilating the former the two-prong dilator of Jackson is valuable because it enables one to pick up the first stricture, dilate it and take the next one and each one in turn.

In dilating the tortuous and tight strictures I like to use the Jackson modification of the Guisez instrument, the flexible filiform on the end of the long steel staff. These are made in dif-

ferent sizes, graduated from the very smallest possible to a fairly large size, there being ten in the set. If the steel staff be perforated just where the filiform is attached to the staff a string can be passed through the hole and a good string-cutting esophagotome is produced, better than the string-cutting esophagotome of Jackson, which is a rigid instrument throughout and in cases like the one reported I have been unable to introduce through the stricture. Of course not much is gained by the use of such an instrument, but every little counts.

The report of the following case is rather typical of the usual run of these cases, pronounced impermeable by the general practitioner and surgeon, but which, to the laryngologist with illuminated instruments, offers abundant opportunity for doing great good to the unfortunate little sufferers.

June 10, 1917, Marshall D., age 3, of Pine Village, Indiana, was brought to my office, because he regurgitated his food and because he was losing flesh. Four months prior to this visit, he swallowed just a thimble full of lye solution, carelessly left within his reach, by his mother after she had cleaned the kitchen sink. Steps were immediately taken to neutralize the poison and seemingly were successful. However, for one month prior to his visit, it was noticed that swallowing was becoming difficult, and regurgitation of food more frequent.

He was immediately sent to St. Elizabeth Hospital for x-ray examination and two sets of plates were made as suggested above. The anterior-posterior plate I show, the other was lost. The latter is the more valuable because it shows the stricture to taper towards the backbone. With the information thus secured we proceeded to treatment. The child was pinned tightly in a sheet and laid on the operating table and held by one of the sisters while my assistant firmly held his head over the head of the table. In this position I passed a fairly large esophagoscope right down to the strictured portion. In looking down the illuminated tube I was able to locate the entrance into the stricture and was able to pass the smallest size Jackson Guisez instrument right into the stomach. The child was not anaesthetized at all, locally or generally. Then at frequent intervals larger and larger sizes were introduced until at the end of six months and after thirty such seances, I was able to pass the largest size, giving the patient ability to swallow with comparative ease, his food well masticated of course. Infrequent dilatations were made for three months longer, with the injunc-

tion to return with the least sign of returning trouble, for so many of these cases need subsequent help at long intervals of time. To date the child has had no further trouble.

At the second seance I attempted to pass Jackson's string-cutting esophagotome but failed. Then it occurred to me to perforate a filiform bougie as above and thus use the string in cutting the stricture. We succeeded admirably.

Such a result is possible in nearly every case of tight stricture of the esophagus. The prognosis in these cases is poor and the mortality high, if the patient is left untreated, or even if gastrostomy is performed for the purpose of making retrograde dilatation which, by the way, is a much more difficult procedure than what it appears to be from the descriptions of the operation at command.

Conclusions:—First: Laws should be enacted and enforced requiring the poison label to be attached to concentrated lye containers, as well as of the various cleansers on the market.

Second: No dilatation of the esophagus should ever be undertaken except under direct inspection.

Third: These cases afford the laryngologist to do a vast amount of good in a comparatively easy fashion.

TWO CASES OF FRACTURED SKULL; WITH SECONDARY MASTOIDITIS, MENINGITIS, AND, IN ONE CASE, BRAIN ABSCESS.

Dr. James Morisset Smith, New York.

The following cases were seen with Dr. William Sharpe at the New York Polyclinic Hospital. Both were fractured skulls involving the base with a secondary mastoiditis, meningitis and in one case a brain abscess.

Case 1. Man, age 43, white, cab driver, family and past history negative.

Present Illness: Fell from the seat of a hansom cab, October 31, 1917. Was picked up unconscious, bleeding from both ears.

Physical Examination: Well developed and nourished, plethoric individual. Unconscious and bleeding from both ears—blood mixed with cerebrospinal fluid. In state of shock—spinal puncture revealed blood under marked pressure. Reflexes exagger-

ated, especially left side. Babinski present in left foot, no convulsions, no twitchings. Ophthalmoscopical. Marked edematous changes—blurring all fundus details and nasal half and its margins. Denoting marked vocal pressure changes.

Operation: By Dr. Sharpe: Left subtemporal decompression November 2; usual incision, temporal muscle incised and retracted: periosteum separated, revealing small bluish black area through lower portion of bone. Bone rongeured, exposing dura under very marked pressure. Latter incised, a clot appearing through opening and bloody C. S. fluid welled out. The clot was subdural—no apparent injury to cortex, excepting vessels which were markedly congested. Clot was removed and wound closed as usual with two drains. Patient left table in good condition.

Remarks: From time of admission except for occasional twitchings about neck, shoulders and arms, unconsciousness marked, with heavy, deep breathing, moves arms and turns to either side. Operation decided upon on account of prolonged unconsciousness, blood in C. S. F. and changes in fundus oculi. T. 102′, P. 94 and R. 18. Patient regained consciousness at 2:30 p. m. (four hours after operation).

Sixteen days later I examined the patient and found a tender post auricular swelling right ear, profuse discharge from canal, neck partially rigid, Kernig sign present and patient stuporous but could be momentarily aroused, spinal fluid cloudy, no bacteria, no report on culture of fluid; temperature ranging from 99½ to 101.

Diagnosis—Mastoiditis and Meningitis. Mastoid Operation.

Exposure of mastoid cortex showed a fracture running below the temporal ridge forward to spine of Henle.

Entire mastoid cavity nicrotic and filled with pus, fracture extending down into the angle between cerebral and cerebellar dura.

Mastoid cells thoroughly exenterated and usual closure and dressing.

Patient died two days later of meningitis.

Case 2. Woman, age 40, white, family history and past history negative.

Present illness dates from an automobile accident October 31, 1917; then was bleeding from right ear at the time of the injury. Patient admitted to Polyclinic Hospital one week later.

Examination showed well developed and nourished patient with ecchymosis of both orbits, mastoid regions, scalp wound 3 inches in occipital region.

Multiple contusions with ecchymosis in neck, trunk and extremities; knee jerk increased, abdominal reflexes absent, no Babinski.

Lumbar puncture showed high pressure and bloody fluid. Pupils slightly enlarged, left larger.

On admission, temperature 101.6, pulse 84.

Ophthalmoscopic: Right eye, mild oedematous changes of the upper nasal quadrant. Left eye, no changes.

Second lumbar puncture, cloudy spinal fluid with symptoms of meningitis.

I saw this patient about three weeks after admission and my examination revealed almost the identical condition in patient above, an unquestionable mastoiditis with marked post auricular tenderness, slight oedema, profuse discharge from canal, mental condition hazy, positive Kernig, neck stiff and cloudy spinal fluid, with a report of no bacteria.

Operation was deferred at this time and the patient observed for two weeks, general condition about the same. Another lumbar puncture was done and the patient showed a slight improvement with the mastoiditis more marked.

At this point I decided to drain the mastoid and instead of doing a complete mastoid operation as in the first case I very quickly exposed the cortex and there was a fracture running forward to the spine of Henle just under the temporal ridge. I then removed the cortex, avoiding the hammer and chisel, and stopped.

The patient immediately began to improve and steadily gained both physically and mentally, until she was finally taken home about two months later, mastoid completely healed.

She showed continuous improvement until February 18, 1918, while in the Neurological Clinic to see Dr. Sharpe, when she developed epileptic seizures, beginning in the face, right arm and leg, then general convulsions lasting one half minute; admitted to the ward for observation, three days no seizures, and again went home.

March 8, 1918, again admitted, showing right facial paralysis with impaired sensation—motor and sensory aphasia weakness with increased reflexes right arm, headache, tenderness over left fronto-

parietal region, normal pulse, temperature, weak lethargic. Consciousness not impaired.

Operation March 18, 1918, by Dr. Sharpe.

Left subtemporal decompression and drainage, usual incision. Dura opaque, thick, non-transparent, not under tension, very little C. S. fluid exuded. Dura adherent to cortex, which was grayish, showing signs of previous meningitis. Cortex did not pulsate.

Left fronto-parietal region explored with a hollow needle. Large abscess, cavity found about 4 cm. from surface of cortex. Large amount of thick, grayish pus was evacuated, showing a pure culture of streptococcus. Usual closure with drainage.

Patient finally died of meningitis three weeks later.

Comment: The above cases bring up some very interesting questions:

First—whether we have a true meningitis in those cases presenting all the clinical symptoms plus a cloudy spinal fluid but no bacteria. Owing to the fact that some of these cases recover opinions differ as to the presence of real meningitis.

Personally I think we do have a bacterial meningitis present but the infection becomes localized and walled off as in these cases, and this accounts for the absence of free bacteria in the fluid as well as recovery in some of the cases, remembering in a diffuse meningitis they die without fail.

These cases showed some improvement following lumbar puncture and while repeated lumbar punctures have been of no avail in cases of diffuse meningitis—when there is evidence of localization it is worth trying as a therapeutic measure.

The prognoses at best in cases of this type are very poor and the decision as to the best thing to do for the patient is a very difficult one; should I again be confronted by a similar situation I should follow the method used in the last case, since we establish drainage by simply removing the cortex and do not interfere with the protecting wall which nature may have thrown around the line of fracture.

45 East 62nd street.

INTERESTING SYMPTOMS IN CONNECTION WITH A CASE OF BRAIN ABSCESS OPERATION AND RECOVERY.

Dr. J. Wesley Bowers, New York.

Mr. F. P.——, age 21, while on a visit at his home over Sunday, came to me for the purpose of having a mastoid wound dressed. He gave the history of having had a primary mastoid operation on the left side two months previous and a secondary mastoid operation by another man one month previous. He complained that some days he felt very badly, at which time he always had a very bad headache. Other days he felt very well. He also said that for several weeks he had been unable to flex his right foot. Later the young man's father stated that his son's disposition had been entirely changed after the first operation. Whereas he had formerly been a very quiet sort of boy, he became hard to control and very unreasonable.

Examination showed a very dirty, granulating wound over the left mastoid with considerable discharge of pus, rough bone at bottom of wound, drum membrane thickened and lusterless, no discharge from the meatus, temperature 100.3, pulse 80. He was absolutely unable to flex his right foot, knee jerks active, no eye symptoms, no errors in pointing, labyrinth normal, hearing 10/20, no aphasia. At this time I advised operation, but it was necessary for him to return to his duties.

He returned one week later and placed himself in my hands. At that time he had severe headache, temperature 101, pulse 82, still unable to flex his right foot.

I did a secondary mastoid operation and found that both previous operations had been very poorly done. There was at least one-quarter of an inch of necrotic bone over the floor of the middle fossa and apparently the antrum had not been entered. There were a considerable number of cells at the tip and a large number of cells in front of and posterior to the sinus. It was a very large mastoid and at both previous operations the operator had simply drained the center. I exposed the middle fossa in the hope of finding some point of entrance of the infection. The dura was absolutely normal in every respect. The sinus was exposed and the dura in the posterior fossa was

exposed. Both were found to be normal. Not having any localizing symptoms, I decided to wait. The first two days following operation the patient's temperature remained at 102, then for a few days it was 101, then dropped to normal. He still complained of headache and after the third or fourth day he began to be stupid. During this time he showed absolutely no other symptoms. On the ninth day post-op he first showed signs of amnesic aphasia, which came on over night. I operated immediately, did a large temperal decompression and removed all the bone over the dura corresponding to the mastoid wound. The dura was perfectly normal in appearance, but under pressure. I made a crucial incision in the dura directly over the mastoid cavity. A grooved directer was passed inward, slightly upward and forward. Nothing was obtained. The directer was then passed directly inward and upward. It had penetrated about one and one-half inches when there was a rush of serum followed by a large quantity of pus filling the large mastoid wound twice. A cigarette drain was inserted into the abscess cavity, and kept in place for two weeks, this being necessary because of the large amount of pus coming from the abscess cavity. The patient immediately began to improve. His headache disappeared, temperature remained normal. It was interesting to note the return of his powers of connecting the two centers. He had completely lost all power of remembering the names of persons, places or things. His memory, however, gradually improved and at the end of two months was apparently normal. He regained forty pounds in weight which he had lost prior to the operation and regained his power to flex his right foot.

There are several striking things about this case. One being the remarkably poor work done at the first two operations. It seemed astounding that such work should be done by any one who is allowed to call himself an aural surgeon. In the next place, it was astonishing that the man should have been allowed to go about with the symptoms displayed with apparently no suspicion of the actual conditions. (On the day he came to my office with a temperature of 101 and a severe headache, he was told that in another week he would be entirely well). Third, it is a question whether under the conditions observed one should immediately go in and look for an abscess, instead of waiting, as was done. I am inclined to think that if I had gone in and had not found the abscess, I would have blamed myself. There

did not seem to be any great risk in waiting, for he had been suffering with the conditions for two months.

Another case I would like to refer to for the sake of information and discussion.

Master A—age 6. One year, six months ago child had had a double mastoid operation performed and the mastoid wounds had been kept open for four months. One month previous he had had influenza-pneumonia with considerable headache and delirium. At this time Dr. Meara had been called in in consultation and had said the child had meningismus. The child apparently became perfectly well and had been up for about one week when it was noticed that he semed to have a temperature. The temperature was found to be 104. The parents had noticed that the child's hearing had been poor for about two weeks. He was prostrated for three or four days, by which time a swelling began to appear in the old mastoid wound on the left side. I was at this time called in consultation. The left mastoid wound was reddened and the scar was apparently swollen slightly. Both membranes were bulging, the temperature was 103. I did a double myringotomy. He was restless and irritable with a temperature between 100 and 103, otherwise there were no other symptoms. The following day I did a secondary mastoid operation on the left side and found it filled with pus and necrotic granulation tissue. That night he became more restless and delirious with projectal vomiting and temperature of 104. In the next eighteen hours he became more stupid and developed spasticity of both feet. No Kernig, no stiffness of the neck. It was decided to explore both mastoid cavities again. The bone was removed over the floor of the middle fossa, the sinus and the posterior fossa on the left side. The dura was normal and under no tension. The right mastoid wound was then opened. The cavity was found to contain a small amount of pus and some necrotic granulation tissue, otherwise it was apparently normal. At this point it was necessary to stop the operation because of the child's poor condition. The child lived six hours longer and died. I do not know what the child had. It did not have meningitis. It must have had a very small abscess near the fourth ventrical.